interventions might reduce caesarean sections moderately if not significantly. We will not know how well the project has worked or whether there will be some delayed impact of having merely published the recommendations until early in 1989. What is already clear is that without clearly targeted initiatives the multifaceted causes driving the rate of caesarean section upwards will influence doctors' behaviour far more than continued calls from researchers for a reduced rate.

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The author receives personal support from the Ontario Ministry of Health, and the work is partly funded by NHRDP grant No 6606-3390-57 from Health and Welfare Canada.

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Anyone for tetanus?

Boosters advised for adults on every decade birthday

An active immunisation programme has meant that tetanus has virtually ceased to afflict children in Britain. In adults there are encouraging downward trends in incidence and death, but about 30-50 cases still occur each year—and between 10% and 60% of these patients die. Half the cases occur in patients over 65, and more than half of these follow gardening injuries; the prognosis worsens with age.¹⁻³ Rare events remain someone else's concern till they strike at home, and Bibby and Dixon's report on the low cover against the disease among their adult patients stems from a death in their own practice (p 598).

Bibby and Dixon have carried out a simple and probably widely reproducible survey of the records of 600 adults attending their surgery. Only 13% were certainly protected against tetanus. Even if the stringency of their judgments was relaxed to fit with the 10 year pattern of booster injections widely recommended on both sides of the Atlantic, around three quarters of their patients were still at risk. This figure is in line with estimates from elsewhere: Grabenstein *et al* reported that 53% of those over 60 reporting for influenza immunisation were inadequately protected against tetanus⁴; and Williams *et al* found that 40-80% were unprotected in their review.³

The theoretical work from which immunisation policies will evolve demands complicated mathematical projections. The standard recent work comes from Denmark, where Simonsen *et al* studied rates of loss of protection under different circumstances.⁵ They report that 28% of patients are unprotected 25-30 years after primary immunisation if no booster has been given. They believe that immunity lasts longer after revaccination than after primary vaccination. They find only one (doubtful) case of tetanus reported in a

patient within five years after receiving primary protection but were not able to comment on whether revaccination on its own at injury guarantees cover. Recognising the risks of hyperimmunisation (neuropathy and anaphylaxis) with too frequent boosting, they advise primary immunisation in infancy, a boost at five years, a further boost at 25 years, and then boosts every 20 years.

This is where theory and practice become uncomfortable bedfellows. Patients' ability to remember their past medical histories is inexact over even short periods of time, and they are unlikely to recollect accurately the dates of injections received more than 10 years previously. The average patient changes doctor about once every 10 years, and the transfer of records between practices means that clinical and administrative information may be lost. It seems wise to prefer revaccination every 10 rather than every 20 years, and arranging this on the decade could add point to the growing fashion of celebrating 40th, 50th, and other similar anniversaries in style. For the patients currently attending the surgery whose immunity is uncertain I recommend a full course for any adult never before immunised (and that means anyone over 40 until proved otherwise) and a single opportunist injection for anyone previously covered but not boosted within 10 years. Once this has been achieved clean trauma need cause no anxiety and dirty wounds need a booster only if the last one was more than five years previously. For those with injuries whose previous immunity is unknown I advise toxoid for all wounds and human tetanus immune globulin as well for dirty ones.

Formulating a biologically appropriate policy is, however, not the only issue: problems in primary care also prevent immunisation of everybody. Firstly, the information overload

that destroyed the informativeness (as against volume) of hospital records in the late 1960s now threatens to do the same to general practice records, and we must support the current pressure being put on practices to have summaries of the health and history of their patients. The ideal format is still to be described, and computers are as likely to add to the problems as to solve them unless the purposes of both the record and the summary are properly thought through.

Secondly, the list of screening, preventive, and educational tasks being allotted to primary care teams is getting out of hand. These teams should take the main responsibility for the early months or years of life, and considerable discussion is taking place over the best way to balance autonomy against intervention in the elderly.⁶ But a more informed discussion is needed on the proper balance (both clinical and economic) between opportunistic and systematic screening and recall systems for people in mid-life. The problem of both the carers and the public forgetting could be minimised by using the decade birthday, which would minimise the risks of patients missing out because of geographical mobility or infrequent use of health services. It would also put a sensible ceiling on what seems likely to become extraordinarily expensive systems of pursuing "non-compliant" patients, who might in

truth end up healthier for having taken responsibility for their own health.

And, thirdly, as the most important negotiations on general practitioner contracts for two decades move to a crucial stage we must warn against the easy attractiveness of measurable but meaningless performance indicators. The philosophy of accountability is not the problem—just the way in which it is to be achieved. Quick solutions will be worse than the problems they aim at preventing.

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Oestrogens and cardiovascular disease

Postmenopausal oestrogens seem to reduce coronary heart disease

The low rates of coronary heart disease in premenopausal women were one justification for an early randomised trial of exogenous oestrogens in the secondary prevention of coronary heart disease in men. The trial was abandoned because the treatment caused more coronary disease.¹ Since the 1970s the deleterious effect of oral contraceptives on mortality and morbidity from cardiovascular disease has been confirmed: the attributable risks are greater in smokers and older women.^{2,3} These findings refer, however, to older preparations and to Western populations with high rates of cardiovascular disease. The risks associated with modern oral contraceptives seem to be lower and are under investigation.^{4,5}

The cardiovascular risks of oral contraceptives are related to both the oestrogen and the progestogen dose through various mechanisms.⁴ The adverse effect of progestogens on high density lipoprotein cholesterol concentrations seems to be an important pathway for increasing the risk of coronary heart disease, and the venous thromboembolic risks are related to the oestrogen dose.^{2,3}

Because of the effects of oral contraceptives researchers initially expected that oestrogens given after the menopause would increase mortality from cardiovascular disease. Because oestrogens are used so widely after the menopause, especially among American women, it is important that their effects should be clarified—even a small effect would be of major importance to public health.⁶ Although published reports on the cardiovascular effects of postmenopausal oestrogens have been confusing,⁷ the recent publication of several prospective studies has clarified matters.

The Framingham study found an increase of half in the risk of cardiovascular morbidity in users compared with non-users but no increase in the total mortality. This study looked at a cohort of 1234 postmenopausal women, a quarter of whom had used conjugated equine oestrogens between 1962 and 1972.⁸ In contrast, the nurses' health study of 32 317 postmenopausal women studied in the late 1970s found an

appreciable protective effect of postmenopausal oestrogens on coronary heart disease; just over half of the nurses had used oestrogens.⁹ The discrepancy between these two studies is probably explained by their different methods: a more rigorous reanalysis of the Framingham data, with harder end points and a modified definition of oestrogen use, found that oestrogens used after the menopause in women aged 50-59 protected against cardiovascular disease; an adverse effect was found in only a few aged 60-69.¹⁰

A well executed study of deaths in a cohort of white women participating in the lipid research clinics prevalence study and seen initially between 1972 and 1976 showed a highly protective effect of non-contraceptive oestrogens on mortality from cardiovascular disease.¹¹ The effect seemed to be mediated through an increase in high density lipoprotein concentrations; it seems unlikely to have been caused by a selection bias for oestrogen use—that is, women at low cardiovascular risk preferentially using oestrogens. The Walnut Creek prospective study found that postmenopausal oestrogens have a protective effect on mortality from all causes, including violent death,¹² which suggests a selection bias. The prospective study with the largest number of hard end points was conducted in a Californian retirement community, and it too found that oestrogens reduced deaths from acute myocardial infarction.⁶ Postmenopausal oestrogens also seem to eliminate the increased risk of coronary heart disease in women who have had bilateral oophorectomy.¹³

Thus the results from all community based prospective studies, except perhaps the Framingham study, show that postmenopausal oestrogens offer substantial protection against the risk of cardiovascular disease. Most of the community based case-control studies have also found a protective effect.⁷ All these prospective studies, however, have been conducted in the United States. Only one pilot case-control study has been reported from Britain,¹⁴ and